



## The Effects of Terminating Creatine Supplementation and Resistance Training on Anaerobic Power and Chosen Biochemical Variables in Male Subjects

by

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*The main objective of this study was to investigate the effects of alkaline creatine supplementation and resistance training termination on anaerobic power and chosen biochemical variables in men. Twenty-three untrained male subjects, which participated in this study, were divided into supplemented (S) (n=13, age 21.4±2.3 y) and placebo (PL) (n=10, age 22.1±2.6 y) groups. The participants from both groups performed resistance training 3 times a week, while subjects from group S were supplemented with alkaline creatine (ACr) for 4 weeks on their training day's with 66.8 mg/kg b.m., and on non training day's with 33.8 mg/kg b.m.*

*To evaluate anaerobic power, the 30s Wingate test was applied. The following variables were registered: relative mean power - RMP (W/kg), relative peak power - RPP (W/kg), time of reaching peak power - TRPP (s) and relative total work - RTW (J/kg). The test was administrated 5 times – before and after 4 weeks of training and supplementation, as well as the first, second and third week after terminating creatine intake and the resistance exercise protocol. Body mass and body composition was also evaluated during the same time span. Blood samples were drawn at rest before the Wingate test for the assessment of IGF-1, hGH, LA and CrN concentration, as well as creatine kinase (CK) and lactate dehydrogenase (LDH) activities. Supplementation with alkaline creatine, combined with a progressive resistance training program, did not significantly influence (ANOVA) the level of RMP (p=0.49), RPP (p=0.31), TRPP (p=0.51), and RTW (p=0.58) in untrained male subjects. In the supplemental group, there was a significant decrease (p<0.01) in TRPP following creatine supplementation and training, yet these values were not significantly different from the control group. The supplementation and training protocol did not influence significantly body mass (p=0.68), yet post hoc analysis indicate a significant increase in body mass (p<0.001) only in group S. The applied supplementation and training protocol did not influence (ANOVA) the concentration of serum CrN (p=0.81), hGH (p=0.26), CK (p=0.49) and LDH (p=0.64) activities. No significant changes were observed in resting blood LA concentrations of the tested subjects. It can be concluded that the ergogenic effect of creatine intake and resistance training was maintained for a week after terminating supplementation and exercise. During the next 2 weeks de-adaptation occurred and most indices of anaerobic power returned to initial values.*

**Keywords:** supplementation, alkaline creatine, resistance training, anaerobic power

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## Introduction

All processes engaged in the growth and metabolism of a cell require energy. The basic source of energy for all living cells is ATP. In muscle cells, ATP is replenished by creatine phosphate (CP), when the concentration reaches 20-30 mM. CP, along with phosphate kinases, creates the phosphagen energy system. Landmark research data confirm the fact that a saturation of phosphates appear during intensive, ATP-dependent, metabolic processes. (Wallmann et al., 1992; Ellington, 2001).

Additionally the CK/CP system plays a very significant role in other energetic processes of the cell, including: buffering of ATP and ADP concentration, preventing local acidosis, functional cooperation of CK with glycolysis, as well as in oxidative phosphorylation.

In 1981, Bessman et al. proposed a phospho-creatine shuttle where Cr and PC play a role of energy transporters, which join sites of ATP synthesis (mitochondria) with sites of energy utilization, rich in ATP-ases (sites of contraction in a muscle cell). Cr and CP are molecules are better transporters of energy than cleaving ATP to ADP, because they are smaller and have a lower negative charge than adenine nucleotides. Creatine and phosphocreatine are often referred together as an energy system, which serves as a temporary energy buffer under conditions of high energy demand (Wallmann et al., 1992). In fast twitch, glycolytic muscle fibers, responsible for fast contractions, the buffering function of the CK/CP system prevails; while in slow twitch, skeletal and heart muscle fibers, the transport function of this system is dominant (Wyss et al., 2000).

Another important function of the CK/CP system is the prevention of ADP cell accumulation, which inactivates cell ATP-ases and causes a loss of adenine nucleotides. During physical activity, declines in adenosine triphosphate (ATP) are prevented when phosphocreatine phosphorylates adenosine diphosphate (ADP) to form ATP. For instance, Hirvonen et al. (1987) measured muscle phosphocreatine in sprinters and found that 88-100% of muscle phosphocreatine was depleted in about 5.5 sec. Phosphocreatine resynthesis is an aerobic proc-

ess that takes approximately 3-6 min to complete, depending on exercise intensity, duration, and the number of bouts. (Dawson et al., 1997).

Because the creatine kinase-phosphocreatine energy system is so critical to maintain ATP levels during exercise, increasing or decreasing basal levels of muscle creatine must alter energy metabolism. The effects of creatine supplementation on muscle function (i.e., strength, endurance, power) have been studied in hundreds of investigations. Ideally, if an individual is able to significantly increase muscle creatine and phosphocreatine levels with supplementation, there is a potential ergogenic effect. Creatine appears to be most effective when exercise time is brief (below 30 s), intensity is maximal and contractions occur over repeated bouts (Branch, 2003). Additionally, creatine supplementation may enhance sprint performance when intense exercise follows or is interspersed during an endurance exercise task (i.e., cycling).

Nutritional supplements and other ergogenic AIDS have gained widespread popularity among professional, amateur/recreational and student athletes for their potential to enhance athletic performance and to provide a competitive edge. Creatine (Cr) has been a popular nutritional supplement among athletes since 1994. A review of literature indicates that many research studies have evaluated the effects of Cr supplementation on muscle physiology and/or exercise capacity in healthy and trained populations (Kreider, 2003; Francuax et al., 2006). Short-term creatine supplementation (e.g., 20 g/d for 5-7 days) has typically been reported to increase total Cr content by 10-30% and phosphocreatine (PCr) stores by 10-40% (Gutiérrez-Sancho et al., 2006). Generally, no study reports a statistically significant ergolytic effort (Izquierdo et al., 2002). Many scientists indicate that long-time resistance training and creatine supplementations disturb the organism's homeostasis. After the end of training and supplementation, it has been observed that homeostasis of the organism returns slowly to pretraining values, yet how long this processes takes is not known.

Thus, the main objective of this work was to evaluate the effects of terminating creatine supplementation and resistance training on anaero-

bic power and capacity, and chosen biochemical variables in male subjects.

### **Material and Methods**

Twenty-three untrained male subjects participated in the study. They were randomly divided into supplemented (S) and placebo (PL) groups. The basic characteristics of the tested subjects are presented in table 1.

#### **Training protocol**

Subjects in both research groups performed similar resistance training 3 times a week with a progressive load increase (3 sets of 12 repetitions and one round of maximal number of repetitions on the basis of which the load value for the next training session was calculated). The initially applied load constituted 60% of the subjects' 1 repetition max (60% 1RM). The rest interval between sets equaled 2 min.

#### **Supplementation**

The S group was supplemented with alkaline creatine (ACr) for 4 weeks on their training days with 66.8mg/kg b.m. in 3 even doses in the morning, 60 min before training and 10 min after the exercise protocol and on non-training days with 33.8 mg/kg b.m. creatine daily. Before the start and after of the experiment, dietary habits of the subjects were collected and analyzed for the energetic values as well as carbohydrate, protein and fat content.

#### **Test**

To evaluate the level of anaerobic power the Wingate test was performed on a Monark 829 ergocycle with MCE v2 (Poland) software. The following variables were registered: time of reaching peak power, relative average power,

relative peak power and total work output. The test was administrated 5 times. Initially, after the 4 weeks of resistance exercise and creatine intake, as well as after the first, second and third weeks of terminating training and supplementation. The research project was approved by the Ethics Committee for Scientific Research at the Academy of Physical Education in Katowice. All of the tested subjects possessed current medical examinations, confirming proper health status and the ability to perform exhaustive exercise

#### **Biochemical parameters**

Resting blood samples were drawn from the antecubital vein at rest before each Wingate test, for the assessment of serum insulin-like growth factor (IGF-1), growth hormone (hGH) concentration as well as creatine kinase (CK) and lactate dehydrogenase (LDH) activities. Serum IGF-1 concentration was determined using the immunoradiometric assay (IRMA) kits and hGH concentration with the use of DSL GH IRMA kits. Serum enzyme activities were assayed by using CK and LDH kits (Randox, UK)

#### **Statistics**

The research results were analyzed statistically by means of two way ANOVA with repeated measures (2 x 5 and 2 x 6) with the use of Statistica 7.1 (StatSoft) software to determine the influence of supplementation and training on anaerobic power, chosen hormone concentrations and enzyme activities in blood serum. When significant differences in F ratio were found, post-hoc Tukey's test was applied. The level of significance for statistical analysis was  $p < 0.05$ .

Basic anthropometric data of the tested subjects		
Variable	Supplemented group ( $\bar{X} \pm SD$ )	Placebo group ( $\bar{X} \pm SD$ )
Age (years)	21.4±2.3	22.1±2.6
Body height (cm)	182.3±5.8	178.3±8.2
Body mass (kg)	74.8±5.4	74.8±8.1

Variables	Group	Training/Supplementation ( $\bar{X} \pm SD$ )				
		Initial	Final	1 <sup>st</sup> week after	2 <sup>nd</sup> week after	3 <sup>rd</sup> week after
Relative mean power (W/kg)	S	8.7±0.5	8.7±0.8	8.8±0.6	8.7±0.6	8.5±0.6
	PL	8.6±0.6	8.5±0.7	8.5±0.7	8.5±0.7	8.4±0.7
Relative peak power (W/kg)	S	10.9±0.9	11.1±1.3	11.2±0.9	11.0±0.7	10.9±1.2
	PL	10.9±0.5	10.7±0.6	10.7±0.5	10.6±0.7	10.6±0.8
Time of reaching peak power (s)	S	7.23±1.55	6.03±0.91*	6.48±1.11	6.86±0.92	6.80±1.18
	PL	7.26±1.39	6.66±1.63	6.89±1.51	7.03±1.68	7.15±1.49
Rel. total work output (J/kg)	S	259.5±15.2	263.1±24.4	265.8±19.3	261.7±15.9	254.8±18.4
	PL	259.1±19.1	257.5±20.2	257.3±19.4	256.1±22.9	254.6±22.5
Body mass (kg)	S	74.8±5.4	76.9±5.6*	76.6±5.5*	76.9±5.7*	76.4±5.5*
	PL	74.9±8.1	75.4±7.9	75.5±7.7	75.3±7.6	74.9±7.7

## Results

### Parameters of the Wingate test

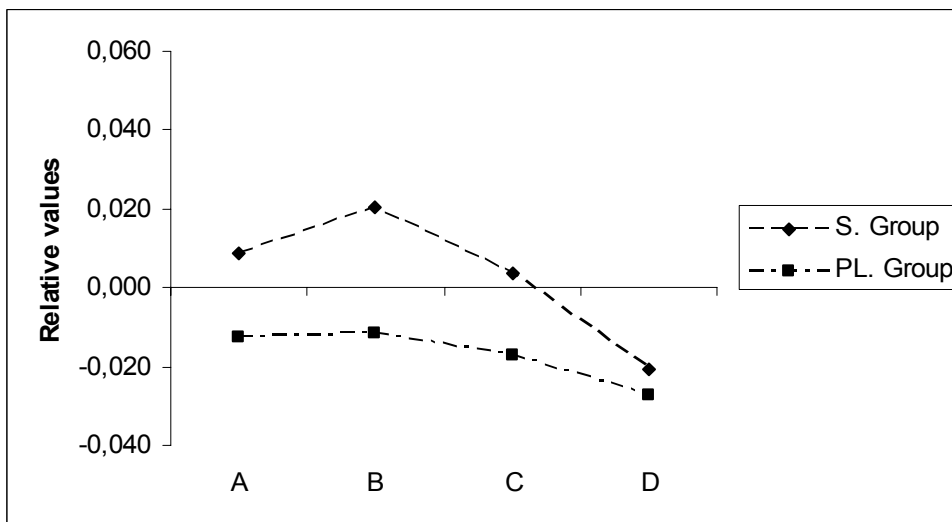
Supplementation with alkali creatine, combined with a progressive resistance training program did not significantly influence (ANOVA) the level of relative mean power (RMP) ( $p=0.49$ ), relative peak power (RPP) ( $p=0.31$ ), time of reaching peak power (TGPP) ( $p=0.51$ ), and relative total work output (RTW) ( $p=0.58$ ) in the male subjects (Table 2).

In the supplemental group there was a tendency for increased RMP, which remained

above initial level for a week from the cessation of creatine intake and resistance training. In the next 2 weeks following termination of the supplementation and resistance training, RMP values were similar to those prior to commencing the experiment (Table 2, Figure 1). In group S there was a marginal increase in RPP, which was maintained for a week after the end of this supplementation and training protocol. As with RMP, in case of RPP during the 2<sup>nd</sup> and 3<sup>rd</sup> week after cessation of the program the values declined to initial level (Table 2, Figure 2). In the supplemental group there was a significant decrease ( $p<0.01$ ) in TGPP following creatine supplementation and training, yet these values

Variables		Training/Supplementation ( $\bar{X} \pm SD$ )					
		Initial	After 2 weeks	Final	1 <sup>st</sup> week after	2 <sup>nd</sup> week after	3 <sup>rd</sup> week after
Creatinine (mg/dl)	S	0.99±0.60	0.79±0.27	1.11±0.36	1.03±0.34	1.26±0.41	1.14±0.39
	PL	1.01±0.64	0.77±0.24	0.93±0.30	1.23±0.81	1.23±0.32	1.22±0.33
hGH (ng/ml)	S	0.82±0.77	0.28±0.43	0.37±0.77	0.50±0.69	1.11±1.19	1.17±1.26
	PL	0.77±0.87	0.30±0.38	0.33±0.45	0.31±0.40	0.37±0.53	1.14±1.16
IGF-1 (ng/ml)	S	374.5±129.0	324.2±110.2	317.8±93.4	336.0±125.1	440.2±191.8	566.8±138.2*
	PL	360.2±122.2	291.8±93.5	300.7±91.5	295.9±43.2	359.6±146.0	531.9±130.2*
CK (U/L)	S	93.6±32.0	237.7±107.2*	275.0±121.0*	140.7±67.1	165.4±65.2	161.1±86.9
	PL	109.4±29.8	181.4±77.1	222.8±126.0	157.8±91.5	163.6±94.3	154.0±72.3
LDH (U/L)	S	272.5±49.0	287.1±72.1	320.5±47.4	317.1±63.4	290.2±37.9	265.9±32.3
	PL	272.8±39.8	293.4±63.3	301.2±71.6	294.4±55.2	289.1±36.7	267.9±49.9
LA (mM)	S	1.46±0.28	2.04±0.27*	2.30±0.70*	2.16±0.48*	1.89±0.53	1.85±0.56
	PL	1.46±0.27	2.55±0.49*	2.61±1.04*	2.30±0.67*	1.98±1.11	1.84±0.79

\*statistically significant in relation to initial values at  $p<0.05$



**Figure 1**

Relative mean power in relation to initial values: A- after 4 weeks of the training and supplementation program, B- after 1 week, C- after 2 weeks, D- 3 weeks after the cessation of creatine intake and training, in supplemented (S) and placebo (PL) groups

were not significantly different from the control group. During the 3 weeks after termination of the supplementation and training program TRPP successively increased (Table 2, Figure 3).

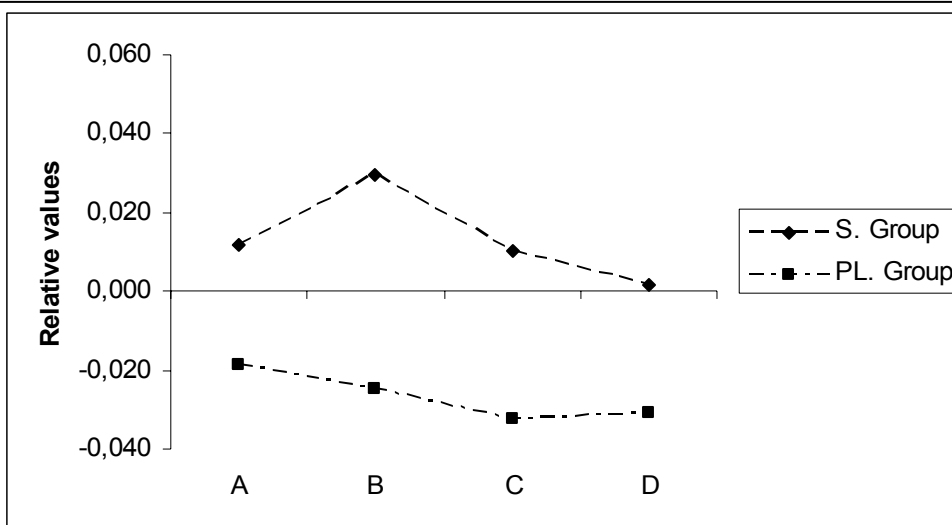
In the S group the highest values of RTW were observed one week after the termination of creatine intake and strength training, yet these values were statistically not different from those observed in the control group (Table 2, Figure 4).

The supplementation and training protocol did not influence significantly body mass

( $p=0.68$ ), yet post hoc analysis indicate a significant increase in body mass ( $p<0.001$ ) only in group S.

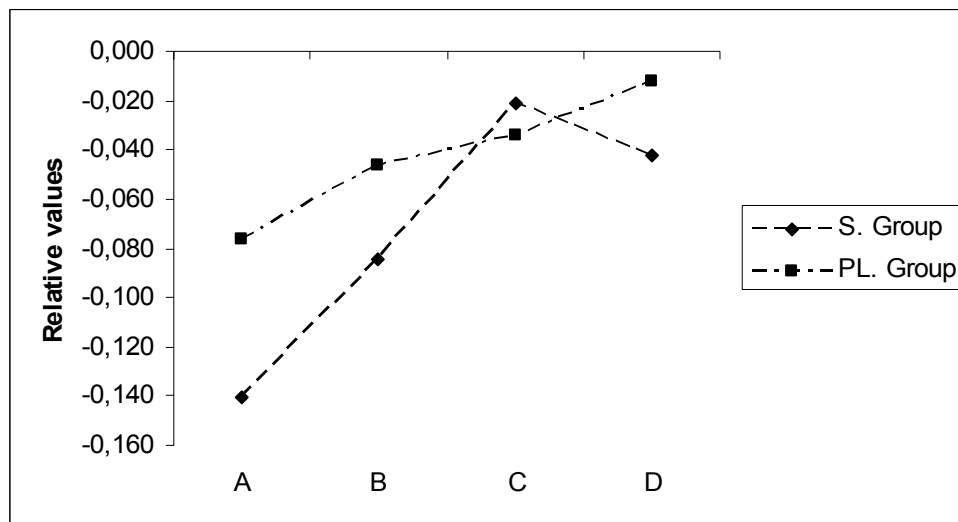
**Biochemical parameters**

The applied supplementation and training procedure did not influence (ANOVA) the concentration of serum creatinine ( $p=0.81$ ), human growth hormone (hGH) ( $p=0.26$ ), creatine kinase (CK) ( $p=0.49$ ) and lactate dehydrogenase (LDH) ( $p=0.64$ ) activities, as well as the blood



**Figure 2**

Relative peak power in relation to base values: : A- after 4 weeks of the training and supplementation program, B- after 1 week, C- after 2 weeks, D- 3 weeks after the cessation of creatine intake and training, in supplemented (S) and placebo (PL) groups

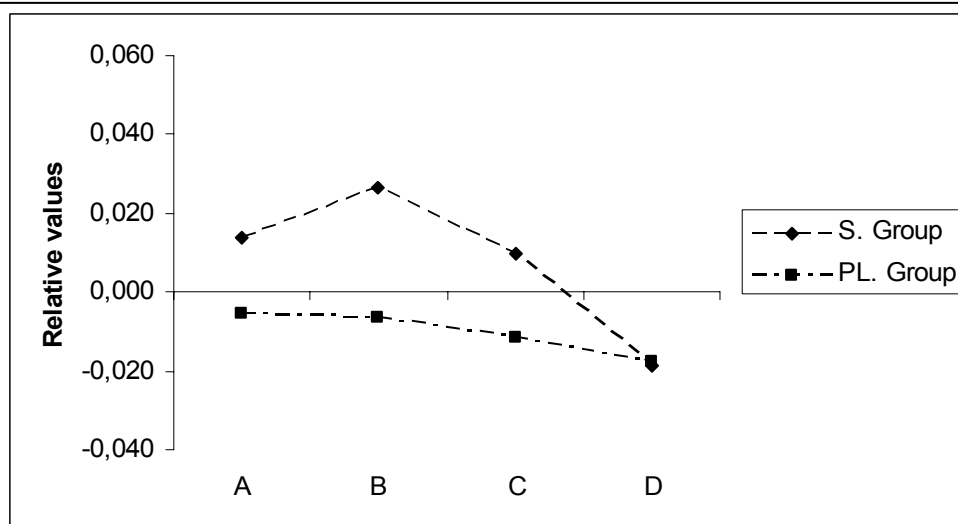


**Figure 3**

Time of reaching peak power in relation to base values: : A- after 4 weeks of the training and supplementation program, B- after 1 week, C- after 2 weeks, D- 3 weeks after the cessation of creatine intake and training, in supplemented (S) and placebo (PL) groups

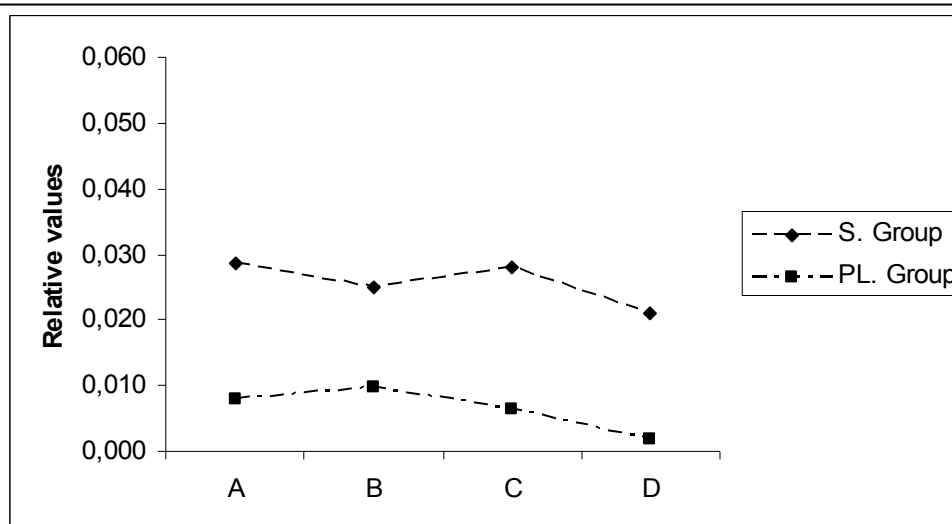
lactic acid (LA) concentration in male subjects. There was a tendency for increased creatinine values in both groups which remained elevated 2 weeks after the cessation of creatine intake and training session (Table 3). During the 2<sup>nd</sup> and 4<sup>th</sup> week of supplementation and training there was a marginal decrease in the serum concentration of hGH, while after the cessation of this protocol a insignificant increase of hGH was observed for 2 and 3 weeks, especially in the supplemented group (Table 3).

Post hoc analysis showed a significant ( $p < 0.05$ ) increase of serum IGF-1 concentration in S and PL groups after 3 weeks of terminating supplement input and training (Table 3). In the supplemental group a significant ( $p < 0.001$ ) increase in CK activity was observed in the 2<sup>nd</sup> and 4<sup>th</sup> week of training, while during the 3 weeks following termination of the exercise and supplementation program CK activity decreased successively, reaching initial values (Table 3). Changes in LDH activity were similar



**Figure 4**

Total work output in relation to base values: : A- after 4 weeks of the training and supplementation program, B- after 1 week, C- after 2 weeks, D- 3 weeks after the cessation of creatine intake and training, in supplemented (S) and placebo (PL) groups



**Figure 5**

Body mass in relation to base values: A- after 4 weeks of the training and supplementation program, B- after 1 week, C- after 2 weeks, D- 3 weeks after the cessation of creatine intake and training, in supplemented (S) and placebo (PL) groups

to those observed in CK. The activity of this enzyme increased insignificantly during the resistance training and supplementation procedures and decreased at a similar rate after the cessation of this program (Table 3).

During the 4 weeks of creatine intake and resistance training there was a significant increase ( $p < 0.05$ ) of LA concentration in both groups. After termination of supplementation and resistance training blood LA concentration decreased successively, reaching basal values (Table 3).

### Discussion

Most research confirm that resistance training programs backed up by daily supplementation with creatine in doses between 10 and 25g allow for significant increases in cell phosphocreatine content (Odland et al. 1996, Yquel et al. 2002). The results of this experiment did not show significant ergogenic effects of creatine supplementation on anaerobic power variables in untrained men, yet increases in all considered variables were much higher in the supplemented group in comparison to the control one. The time needed for reaching peak power decreased significantly following the 4-week training and supplementation protocol ( $p < 0.01$ ).

The new aspect of this research is related to the evaluation of power and biochemical variables after termination of the supplementation

and resistance program. In general it can be stated that the ergogenic effects of creatine intake and resistance training were maintained for a week after cessation of the exercise and supplementation protocol. During the 2<sup>nd</sup> and 3<sup>rd</sup> week after terminating the program most anaerobic power variables slowly declined, reaching initial values at the end of the 3-week detraining period.

The review of literature regarding creatine supplementation and possible ergogenic effects gives conflicting results. For example, Levesque et al. (2007) attempted to determine if creatine supplementation (20g creatine/d, for 6 days) would enhance repetitive cycling sprint performance with near LT activity between sprinting bouts. The results suggest that creatine supplementation involving repetitive sprints is not effective when intense activity occurs between sprinting bouts and would therefore not be recommended for such competitors. Jäger et al. (2008) evaluated the effects of oral creatine pyruvate and creatine citrate supplementation on exercise performance in healthy young athletes after 28 days of creatine-pyruvate (5g/d) or creatine citrate (5 g/d) intake. The tested subjects performed ten 15 s exercise intervals, each followed by 45 s rest periods. It was concluded, that four weeks of creatine+pyruvate and creatine+citrate intake significantly improves performance during intermittent handgrip exer-

cise of maximal intensity and that creatine-pyruvate might benefit endurance, due to enhanced activity of aerobic metabolism. Rawson et al. (2003) reported, that creatine supplementation and concurrent resistance training results in an 8% increase in strength and a 12% increase in muscular endurance, in comparison to resistance training alone. It could be hypothesised that chronic creatine supplementation does not have a direct effect on skeletal muscle (e.g. protein synthesis) but simply enhances the ability of athletes to train more intensively (e.g. complete more repetitions of each exercise, faster recovery between sets etc.), via increased basal muscle phosphocreatine and glycogen, and faster phosphocreatine resynthesis. In this manner, creatine supplementation acts as a training aid, by allowing athletes to train at higher volume/intensity over time. Evidence to support this includes spontaneously higher training volumes in creatine vs placebo subjects during a 12-week resistance training intervention (Volek et al. 1999, Kreider 2003). Additionally, others have found that when training volume is controlled for through voluntary or electrically-stimulated contractions (Stevenson et al. 2001), there is no apparent effect of the creatine. Thus there is evidence to support the contention that muscular adaptations to creatine supplementation are dependent on increased training loads. However, Arciero et al. (2001) showed that chronic creatine ingestion (20g/d for 5 d followed by 10g/d for 23 d), with or without resistance training, results in significant increases in bench press (creatine without any training and creatine plus resistance training at 8% and 18% improvement, respectively); and leg press (creatine with no training and creatine plus resistance training at 16% and 42% strength increases, respectively). Eckerson et al. (2008) determined the effect of 30 days of single dose creatine supplementation with phosphate salts (5g Cr + 4g phosphate) on body weight and anaerobic working capacity in men. They suggested, that combined supplementation with creatine and phosphate salts increase body weight but was not effective for increasing anaerobic working capacity in men. Izquierdo et al. (2001) indicate that the most popular and effective method of supplementation consists of acute creatine loading over a

short period of time (5 to 7 days). This has commonly been achieved by administering 20-25 g/day of creatine monohydrate, divided into 4-5 doses of 5 g each, over 5-7 days. Such protocols have been shown to significantly increase muscle creatine content and to improve performance in high intensity, intermittent exercise). Zajac et al. (2004) showed that both, short-term (6day/15g) and long-term supplementation protocols (30day/5g) are equally effective in improving anaerobic power and capacity in well-trained basketball players.

Currently, three theories exist that explain how creatine intake improves performance during anaerobic exercise. First, it is mentioned that following Cr supplementation the total Cr and phosphocreatine (PCr) levels would be higher and consequently more potential energy would be available; that is to say, greater ATP concentration during anaerobic exercise of very short duration (e.g. <30 s) (McConnell et al. 2005). Secondly, the improvement in physical performance in intermittent anaerobic exercise is due to changes in the rate of PCr resynthesis during rest periods between intensive bouts of exercise (Smith et al. 1998). The last theory indicates that Cr use could have the potential to diminish lactate accumulation by buffering H<sup>+</sup> ions produced during glycolysis (Pluim et al. 2006).

In this research the results of ANOVA did not show a significant effect of creatine intake and resistance training on body mass, yet post hoc tests showed a significant increase in body mass (2.8%) only in the supplemental group, which was maintained throughout the 3-week detraining period.

One of the purported side effects of oral creatine supplementation regularly mentioned by consumers is increased total body mass, particularly muscle mass. Indeed the average increase in body mass reported in the literature varies from 1 to 2 kg or 1-2.3% of total body mass (Terjung et al. 2000). Nevertheless, about 30% of published articles do not report any changes in body mass (McConnell et al. 2005). The increase of body mass might be a result of water retention in muscle tissue or an increase in dry matter, possibly glycogen and proteins. Nonetheless, the mechanisms by which creatine supplementation could increase intracellular water remain unclear. Creatine is a highly polar



molecule that plays a major role in regulating osmolarity in the cells. Without exchange of other ions, an increase in creatine content should increase cellular osmolarity and consequently, intracellular water volume. Another explanation involves a higher glycogen content (about 25%) observed when creatine supplementation is combined with resistance training (Derave et al. 2003). While stored in skeletal muscle, 1 g of glycogen is accompanied by 2 to 3 g of water, which could explain an increase in muscle water content of about 300 g.

Creatine intake combined with a progressive resistance program did not influence significantly the chosen biochemical variables: creatinine, human growth hormone (hGH), insulin-like growth factor-1 (IGF-1), creatine kinase (CK), lactate dehydrogenase (LDH) and lactate (LA). The serum level of creatinine decreased insignificantly after 2 weeks of creatine intake and then slightly increased. This level was maintained during the 3-week period of terminating supplementation and training. The level of IGF-1 increased in both groups after the 3-week detraining period, while no changes were observed after the 4-week resistance program accompanied by creatine intake. It can be suggested that most of the biochemical variables were affected by the resistance exercise and not the creatine intake. There were significant increases in creatine kinase activity throughout the 4-week supplementation and exercise protocols, followed by a decrease of this marker to initial values during the 3-week detraining period. Similar tendencies were observed in regards to LDH. The resting concentration of plasma LA increased successfully during the resistance training period and was significant in both groups. There is some evidence that most of these biochemical changes are caused by resistance exercise not creatine intake. Based on this fact, because creatine supplementation results in a rapid increase in body mass and fat-free mass, it has been hypothesised that creatine induces hypertrophy through endocrine mechanisms. Op't Eijnde et al. (2001) examined the combined effects of resistance exercise in creatine-loaded subjects (20 g/d for 5 days) and found that the growth hormone response to exercise was unaltered by creatine. Schedel et al. (2000) however, found increased growth hor-

mon levels (83%) in response to a 20 g oral creatine intake. It is difficult to resolve the practical application of these data, as the increase in growth hormone concentrations were similar to what is seen following exercise, and athletes do not typically ingest 20 g of creatine per serving. These available data indicate that creatine supplementation (20-25 g/d for 5-7 days) as it is ordinarily practiced by athletes, does not alter exercise responses to testosterone, cortisol and growth hormone. Thus it seems unlikely that increases in body mass and fat-free mass secondary to creatine supplementation are hormonally mediated. The fact that a large unaccustomed dose of creatine (20 g/serving) can increase growth hormone concentrations requires further investigations (Schedel et al. 2000).

Santos et al. (2004) reported a blunted increase in plasma creatine kinase (19%) and a substantial increase in plasma lactate dehydrogenase (100%) in creatine supplemented athletes following a 30 km run. Rawson et al. (2001) found no attenuation of creatine kinase, lactate dehydrogenase, range of motion, soreness, or strength following 50 maximal eccentric contractions of the elbow flexors or a high-repetition squat test (15 to 20 reps at 50% 1 RMAX). Currently, there is insufficient data to claim that oral creatine supplementation reduces muscle damage or enhances recovery from stressful exercise, but these studies do indicate that creatine supplementation does not further increase muscle damage as has been suggested by some authors. Deldicque et al. (2005) investigated the effect of creatine supplementation on insulin like growth factor (IGF-1 and IGF-2) mRNA expression including the PI3K-Akt/PKP-mTOR-signaling pathway, in adult human skeletal muscle. IGF-I and IGF-II mRNA were slightly, but significantly, increased after creatine supplementation (5 days, 21 g/d). Also resistance exercise was shown to increase both IGF-I and IGF-II mRNA, yet creatine did not potentiate this effect.

## Conclusions

The 4-week alkaline creatine supplementation procedure (66.8 mg/kg b.m./d on training days and 33.8 mg/kg b.m. on off days) did not significantly affect the level of anaerobic power evaluated by the 30s Wingate test. The applied

supplementation and exercise procedure allowed for maintaining slightly elevated values of anaerobic power and working capacity for a week after terminating exercise and creatine intake. The deadaptive processes began about a week after the cessation of exercise and supplementation, which was confirmed by a return of most variables to initial values during the 2<sup>nd</sup> and 3<sup>rd</sup> weeks of detraining. The chronic creatine intake and resistance exercise also did not significantly affect the activity of CK and

LDH, potential markers of muscle cell damage. hGH, IGF-1, creatinine, as well as rest LA concentration, were also not influenced by the supplementation and exercise procedures. It can be suggested that in untrained men, deadaptation begins a week after terminating creatine and resistance training; thus, in order to maintain increases in anaerobic power and body mass, training and supplementation must be resumed.

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